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Technical note

# Hemodynamic phenotyping 4.0

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ABSTRACT

The concept of integrating hemodynamic variables to define specific profiles or phenotypes has been established for decades. Describing hemodynamic phenotypes plays a key role in educating healthcare professionals about cardiovascular physiology, enhancing the understanding of shock mechanisms, and informing treatment strategies. Recently, two notable innovations have emerged to support bedside identification of hemodynamic phenotypes: machine learning (ML) algorithms and visual decision support tools. When it comes to "small data," such as a limited set of hemodynamic variables, ML algorithms may not be essential for data integration or interpretation. In addition, the hemodynamic phenotypes identified by ML techniques often mirror traditional textbook profiles, though occasionally with inconsistencies that may impact patient safety. This raises valid questions about the need to integrate complex and proprietary ML algorithms for bedside hemodynamic assessment. By contrast, visual tools leverage clinicians' innate ability to process graphical information rapidly, improving the understanding of cardiovascular physiology and enabling recognition of hemodynamic profiles at a glance. As such, they may offer a practical, accessible, and cost-effective alternative to ML-based solutions. Future studies comparing the clinical impact of visual *versus* ML-driven phenotyping are now needed to guide further development and implementation.
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The concept of integrating hemodynamic variables to define specific profiles or phenotypes has been established for decades. As early as the 1970s, anesthesia and critical care textbooks featured detailed tables describing these phenotypes based on measurements from pulmonary artery catheters. For instance, hypovolemic shock is typically marked by reduced cardiac output, stroke volume, and cardiac filling pressures (right atrial and pulmonary capillary pressures). Cardiogenic shock, on the other hand, presents with low cardiac output and elevated filling pressures and systemic vascular resistance. In contrast, distributive shock is usually characterized by preserved or increased cardiac output and reduced systemic vascular resistance. Today, similar hemodynamic phenotypes can be identified using more modern cardiac output monitoring techniques, such as transpulmonary thermodilution and pulse contour analysis.

Describing hemodynamic phenotypes plays a key role in educating healthcare professionals about cardiovascular physiology, enhancing the understanding of shock mechanisms, and informing treatment strategies. Recently, two notable innovations have emerged to support bedside identification of hemodynamic phenotypes: machine learning (ML) algorithms and visual decision support tools. Both are explored in this technical note.

## 1. Machine learning algorithms

Artificial intelligence (AI) refers to the development of systems or machines capable of mimicking human intelligence to perform tasks such as learning, reasoning, problem-solving, and decision-making [1]. Machine learning, a subset of AI, involves algorithms that enable computers to identify patterns in data and improve their performance over time [2,3]. With advances in computing power and ML techniques, it is now possible to analyze large volumes of data rapidly, including in real-time at the bedside [3,4].

Geri *et al.* [5] applied an ML clustering method using clinical and transesophageal echocardiographic data to investigate cardiovascular phenotypes in 360 patients with septic shock. The algorithm

*Abbreviations:* AI, Artificial Intelligence; ML, Machine Learning; ICU, Intensive Care Unit.

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identified five distinct phenotypes: hyperkinetic (n = 84), right ventricular failure (n = 81), hypovolemia (n = 70), left ventricular systolic dysfunction (n = 64), and patients well resuscitated (n = 61). As expected, intensive care unit (ICU) mortality was highest (50%) in patients with left ventricular systolic dysfunction and lowest (21%) in those who were well-resuscitated. The authors noted that the potential utility of such clustering for optimizing hemodynamic support at the bedside remains to be determined. Similarly, Guinot *et al.* [6] employed an ML clustering approach incorporating clinical, biological (NT-proBNP), and echo-Doppler data to identify phenotypes of fluid overload in 145 ICU patients. Their analysis yielded three phenotypes: congestion with moderate alterations of ventricular function (n = 75), congestion with preserved cardiac function (n = 50), and congestion with severe biventricular dysfunction (n = 20). Once again, patients in the group with severe dysfunction had worse outcomes, including higher rates of mortality, acute kidney injury, and prolonged ICU and hospital stays. However, whether clinicians presented with the same data would classify patients similarly, and whether such classification would influence treatment decisions, remains unclear.

In a pilot study, Kouz *et al.* [7] also employed a clustering approach to explore the hemodynamic mechanisms underlying intraoperative hypotension and identified six distinct phenotypes. These were labeled as myocardial depression, bradycardia, vasodilation with increased cardiac index, vasodilation without increased cardiac index, hypovolemia, and mixed types. The authors suggested that automating hemodynamic phenotyping with AI may help reduce the cognitive burden associated with integrating and interpreting hemodynamic data. In a very similar study involving surgical and critically ill patients and exploring hemodynamic phenotypes related to hypotension, Jian *et al.* [8] used an unsupervised deep learning model. This time, their model derived four distinct phenotypes, labeled vasodilation, bradycardia, hypovolemia, and cardiac dysfunction. These findings emphasize that the identification of hemodynamic phenotypes depends on the ML model used, even though for any given hypotensive event, there is only one physiologic "ground truth" and likely one optimal treatment approach.

Machine learning offers the clear advantage of rapidly processing large datasets, which can be valuable in complex clinical situations [1,2]. However, understanding the mechanisms behind hemodynamic instability often requires only a few key variables (*e.g.*, vasodilation = low blood pressure with preserved cardiac output). Moreover, ML algorithms may make classification errors, or to be more specific, labeling may not reflect the real physiologic status. For instance, in the above-mentioned studies by Kouz *et al.* [7] and by Jian *et al.* [8], some patients were labeled as having "bradycardia" despite heart rates >60 bpm, or as "vasodilation" despite normal or elevated vascular resistance [9]. If such algorithms are used to guide therapy, misclassification could lead to inappropriate treatment, such as administering chronotropic agents to patients with normal heart rates or vasopressors to those without true vasodilation. These issues raise serious concerns about both patient safety and medical liability [3,9]. Who is responsible in case of misdiagnosis or adverse outcome—the developer or the clinician? Such concerns will likely increase with AI-driven closed-loop systems that not only suggest treatment options but also deliver treatments automatically [3,10].

Another application of ML methods is known as reinforcement learning. It enables algorithms to learn how to behave in an environment by interacting with it and receiving feedback in the form of rewards or penalties. In medicine, it can be used to identify treatment pathways associated with the best possible outcome. For instance, Komorowski *et al.* [11] used a large ICU database (containing more than just hemodynamic data) to train a reinforcement learning algorithm that determines when to administer fluids or vasopressors, and in what doses. The reward was 90-day survival. The algorithm recommended strategies different from human doctors, such as giving less fluid in some cases, or starting vasopressors earlier in others. When tested retrospectively, patients whose actual treatment matched the algorithm recommendations had significantly lower mortality. In the more recent OVISS trial, Pirracchio *et al.* [12] developed a reinforcement learning model for initiating vasopressin in ICU patients who were already receiving norepinephrine. The model suggested starting vasopressin in more patients (87% *vs.* 31%), earlier, and at lower norepinephrine doses compared to real-world clinical practice. In large validation datasets, the model demonstrated a significant mortality reduction: when clinicians' actions aligned with the algorithm rule, the adjusted odds ratio for in-hospital mortality was 0.81 in favor of the reinforcement learning-guided strategy. Of note, these approaches are not designed to describe or identify specific hemodynamic phenotypes but to support therapeutic decisions by refining timing and dosage in a way that may outperform average clinician practice. They deserve further investigations in prospective and real-life conditions.

## 2. Visual decision support tools

Traditionally, hemodynamic variables are shown as separate metrics in a tabular format, often distinguished by color but offering little emphasis on their physiological relationship or clinical significance. This forces clinicians to mentally synthesize the data, a process that can be time-consuming and error-prone [10]. Interestingly, the human brain processes visual information significantly faster than text [10], and multiple studies have shown that graphical displays improve the speed and accuracy of change detection while reducing cognitive workload [13–16]. Gurushanthaiah *et al.* [13] showed that graphic displays may enhance the detection of acute changes in patient physiologic status during anesthesia administration. Blike *et al.* [14] demonstrated that anesthesiologists using a graphical display committed significantly fewer diagnostic errors when interpreting physiologic data. For instance, the diagnosis of shock etiology was completed more rapidly when the graphical display was used. In a simulation study, Albert *et al.* [15] observed that the use of a graphical display reduced the time to detection and the time to treatment of cardiopulmonary adverse events. In a systematic review of 31 studies, Gorges and Staggers [16] found that all but one study reported significant differences between traditional numerical displays and graphical displays. Eighteen studies reported a faster detection of adverse events, 13 studies an improved accuracy in clinical decision or diagnosis, and 3 studies a decrease in mental workload.

Over the past 15 years, hemodynamic monitors have increasingly incorporated cockpit-style displays featuring color-coded dials (*e.g.*, green, yellow, and red) to flag deviations from normal ranges or therapeutic targets [10,17]. A logical next step is to organize these visualizations based on physiological interdependencies [17]. For example, mean arterial pressure results from cardiac output and systemic vascular resistance; cardiac output, in turn, depends on stroke volume and heart rate. Additionally, since most anesthetic agents reduce vascular tone, depth of anesthesia plays a critical role in determining systemic vascular resistance. Visualizing these relationships, as illustrated in Fig. 1, could allow clinicians to instantly recognize hemodynamic profiles, facilitating timely and accurate interventions. In essence, by translating hemodynamic data into an intuitive visual format, this approach may offer a simpler yet equally effective alternative to ML-based decision support systems [9,17].

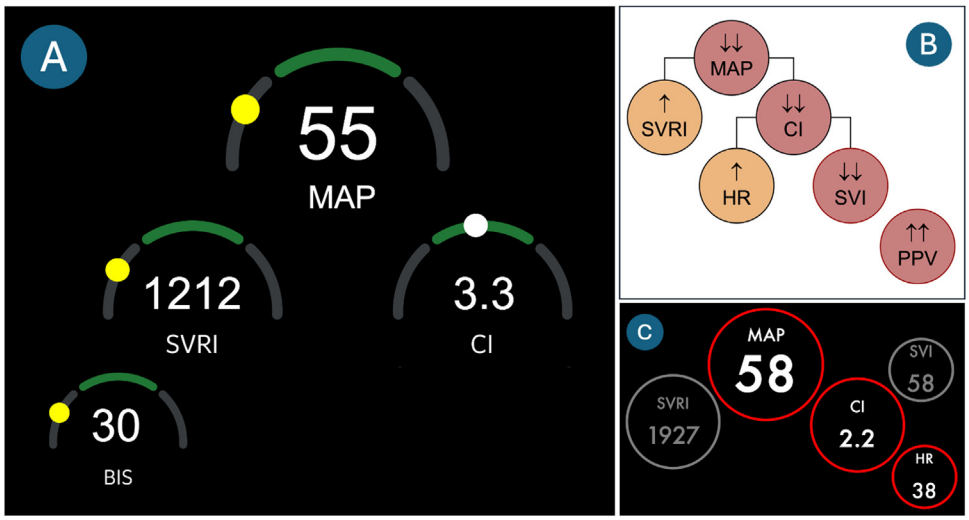


**Fig. 1. Hemodynamic phenotyping at a glance.** Examples of patterns enabling clinicians to visualize the underlying mechanisms of hypotension.
Example A: hypotension related to vasodilation and excessive administration of anesthetic agents; Example B: hypotension related to a decrease in blood flow in a fluid responder patient; Example C: hypotension related to a decrease in blood flow because of bradycardia.
Abbreviations: BIS = Bispectral index, CI = cardiac index, HR = heart rate, MAP = mean arterial pressure, PPV = pulse pressure variation, SVI = stroke volume index, SVRI = systemic vascular resistance index.

## 3. Conclusion

In summary, despite the growing enthusiasm around AI-enabled tools, a clear gap remains between their perceived promise and demonstrated clinical utility. When it comes to "small data," such as a limited set of hemodynamic variables, ML algorithms may not be essential for data integration or interpretation. In addition, the hemodynamic phenotypes identified by ML techniques often mirror traditional textbook profiles, though occasionally with inconsistencies that may wrongly impact therapeutic decisions.

This raises valid questions about the need to integrate complex, proprietary, and costly ML algorithms for bedside hemodynamic assessment. By contrast, visual tools leverage clinicians' innate ability to process graphical information rapidly, improving the understanding of cardiovascular physiology and enabling recognition of hemodynamic profiles at a glance. As such, they may offer a practical, accessible, and cost-effective alternative to ML-based solutions.

Whatever the solution—whether visual or ML-driven—an in-depth understanding of cardiovascular physiology and of the limitations of current cardiac output monitoring tools remains essential. These systems should be regarded for what they are: aids to support decision-making, not substitutes for clinical reasoning in a given scenario. Future studies assessing the clinical impact of visual *versus* ML-based phenotyping will be key to informing their further development and implementation.

## Ethics approval and consent to participate

Not applicable.

## Consent for publication

Not applicable.

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None.

## Data availability

Not applicable.

## Declaration of competing interest

FM is the founder of MiCo, a consulting and research firm based in Switzerland. MiCo does not sell any medical device, and FM does not own shares from any medtech company. OAA has a consulting role for BD Advanced Patient Monitoring, Vantive, and Medtronic.

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